Fusiform Lenticulostriate Artery Aneurysm with Subarachnoid Hemorrhage: The Role for Superselective Angiography in Treatment Planning

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Summary

Aneurysms of the lenticulostriatal perforating arteries are rare and either involve the middle cerebral artery-perforator junction or are located distally in basal ganglia. We describe a rare ruptured fusiform lenticulostriatal perforating artery aneurysm arising from a proximal M2 MCA branch, discerned on superselective microcatheter angiography, presenting solely with subarachnoid hemorrhage (SAH).

A 50-year-old previously healthy man presented with diffuse SAH and negative CT angiogram. Cerebral angiogram demonstrated a 2 mm fusiform aneurysm presumably arising from the right lateral lenticulostriate perforator but the exact origin of the perforator was unclear. Superselective angiography was required to precisely delineate the aneurysm and its vessel of origin and directly influenced treatment planning (surgical trapping).

Superselective microcatheter angiography provides both an option for endovascular therapy as well as more accurate delineation for surgical planning for these rare aneurysms.

Introduction

Aneurysms of the lenticulostriatal perforating arteries (LSA) are rare and most often involve either the junction of a perforator with the middle cerebral artery (MCA) trunk or are distally located in the vessel within the basal ganglia (BG) and frequently present with neurological deficits such as hemiparesis and aphasia [1-4]. LSA aneurysms are often associated with tumors or vascular diseases such as arteriovenous malformations, moyamoya disease, hypertension and systemic lupus erythematosus vasculitis [2,3,5]. Treatment of LSA aneurysms is often technically difficult due to either the intraparenchymal location or the intimate relationship with adjacent lenticulostriate perforators. Both surgical and endovascular treatments have been described [2-7]. We describe a rare ruptured fusiform LSA aneurysm arising from a proximal M2 MCA branch, discerned on superselective microcatheter angiography, presenting solely with SAH. Superselective angiography was required to precisely delineate the aneurysm and directly influenced treatment planning.

Case Report

A 50-year-old previously healthy man presented with sudden onset headache and nausea without neurological deficit. Emergent computed tomographic (CT) scan demonstrated diffuse SAH, more pronounced on the right, and mild hydrocephalus (Figure 1A). CT angiogram was normal (Figure 1B,C). A cerebral angiogram demonstrated a small, approximately 2 mm fusiform aneurysm posterosuperior to the mid right M1 segment, presumably arising from a lateral lenticulostriate perforator (Figure 1D). Exact origin of the perforator was unclear, despite a 3D spin angiogram (image not

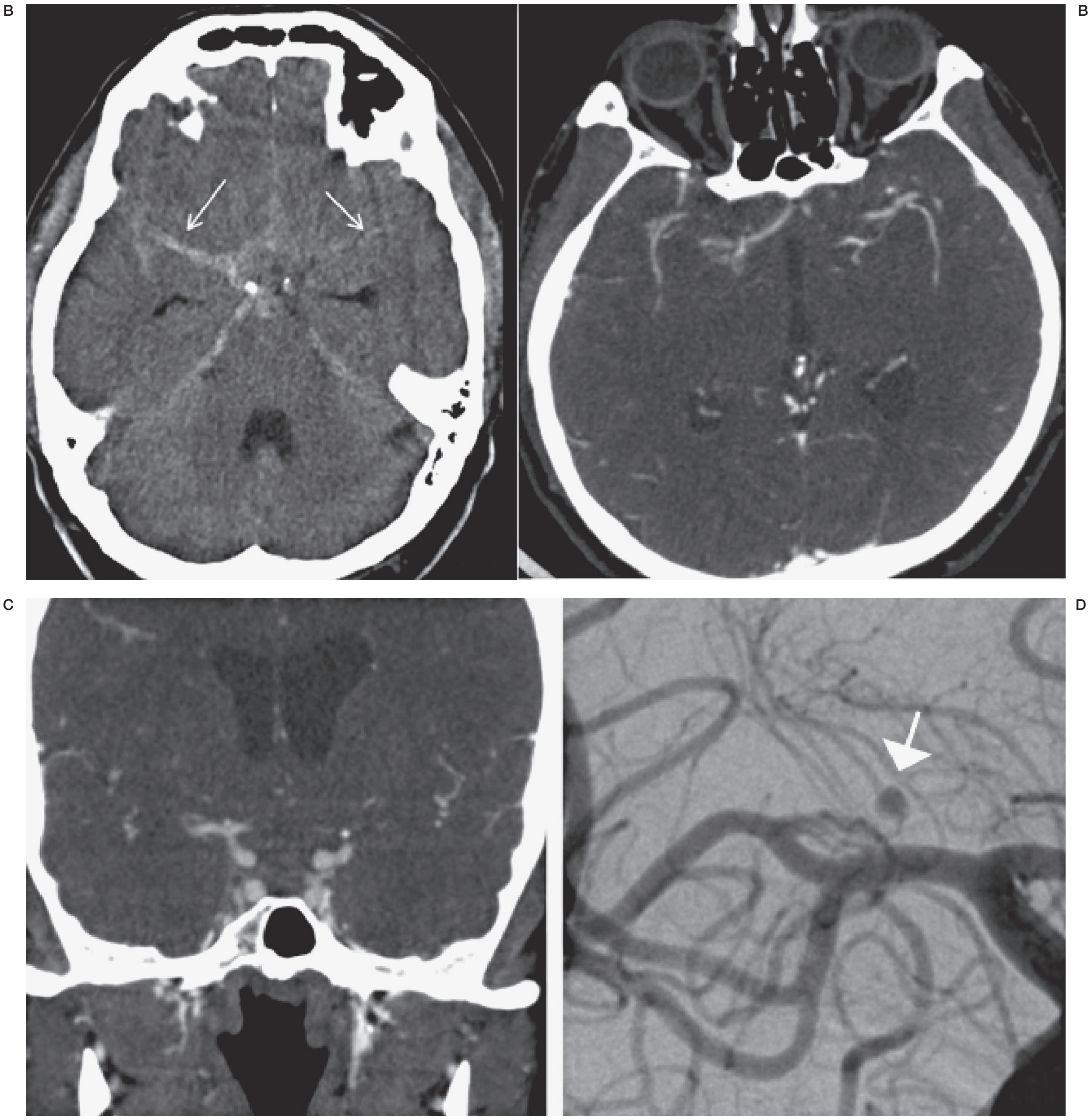

Figure 1 A) Noncontrast head CT scan demonstrating diffuse SAH (white arrows) with blood predominantly on right side. B,C) Axial and coronal CT angiogram did not reveal the aneurysm. D) Anteroposterior view of right internal carotid selective digital subtraction angiography demonstrating lenticulostriate fusiform aneurysm (thick white arrow). Exact vessel origin could not be determined despite 3D spin angiogram (not shown).

shown). There was no evidence of underlying vascular disease or additional aneurysms. An angiogram was repeated after five days with no change in the aneurysm. Preoperative MRI was non-contributory. Superselective angiography through a microcatheter with the patient under general anaesthesia revealed the 2 mm fusiform aneurysm arising from proximal aspect of lateral LSA arising from a proximal right M2 branch coursing medially (Figure 2). We were unable to obtain a stable microcatheter position in this vessel due to the sharp angulation at its origin and potential endovascular therapy was abandoned. This procedure confirmed the vessel of origin, however, and the lateral nature of this vessel, along with the CT showing only

SAH but no parenchymal hemorrhage, made it much more probable that the aneurysm was entirely within the subarachnoid space, and thus more easily surgically accessible. Patient proceded to a frontotemporal craniotomy and two surgical clips were used to trap the aneurysm (Figure 3).

Postoperatively, the patient had mild left facial weakness and diplopia and CT demonstrated a small right basal ganglia infarct (Figure 4A). Clinical symptoms resolved after few days. Check cerebral angiogram on third post operative day, showed no residual aneurysm or vasospasm (Figure 4B).

Discussion

Lenticulostriatal perforating artery aneurysms are rare. They are seen predominantly in association with vascular diseases, such as moyamoya disease, arteriovenous malformations, hypertension associated microaneurysms, systemic lupus erythematosus vasculitis and with tumours [2,3,5]. Most LSA aneurysms reported in literature are located in the distal course of the artery, within the basal ganglia. Proximal lenticulostriatal aneurysms are most often MCA-LSA junction aneurysms, involving some part of the MCA trunk, and are not true LSA aneurysms [5,8,9].

Most ruptured LSA aneurysms present with intraparenchymal haemorrhage (IPH). In an analysis of the literature on ruptured LSA aneurysms, Sakai et al.[4] reported that 11 out of 12 patients with ruptured LSA aneurysms had CT evidence of basal ganglia IPH. Gandhi et al., in a series of six patients with seven LSA aneurysms (six ruptured, one unruptured) reported basal ganglia, intraventricular and subarachnoid hemorrhage, with one patient having solely SAH [10]. All the above patients had one of the predisposing factors, which included hypertension, moyamoya disease or cocaine abuse.

The pathobiology of these vascular abnormalities relative to LSA has been debated. Charcot and Bouchard [11] both postulated that ruptured microaneurysms, also referred to as miliary aneurysms, of perforating arteries were responsible for hypertension-related BG hemorrhages [11,12]. These miliary aneurysms located on the LSA were thought to be associated with atherosclerosis and lipohyalinosis of small caliber vessels, similar to that of the LSA. Despite their presence been confirmed in postmortem angiograms, others have questioned this hypothesis because of the low incidence of true microaneurysms in patients with BG IPH. Instead, pseudoaneurysms, dissecting aneurysms, and weakened vessel walls with lipohyalinosis have been observed in evacuated hematomas supplied by LSA [3]. Another important etiological factor in the formation of LSA aneurysms is thought to be moyamoya or moyamoya-like disease, which often involves stenosis or occlusion of a major intracranial vessel, and the formation of ganglionic collaterals. Approximately, 1.5-12.9% of these collateral vessels are associated with aneurysm formation [6].

Aidin et al. [13] analyzed intraoperative anatomical findings of the LSA in 60 patients with MCA aneurysms, 85 % of LSAs (n = 204) arose from the M1 segment, and 15% (n = 36) arose from proximal part of an M2 branch. However, angiographically, they noted that the recurrent course of lateral perforators along their parent vessels may give false impression that they arise more medially than is actually true. This elucidates the usefulness of superselective microcatheter angiography to delineate the exact origin of LSA relative to MCA. This can be extremely important for the neurosurgeon in localizing the culprit vessel intraoperatively and hence, minimizing unnecessary exploration and injury to lenticulostriatal perforators.

Definitive treatment of LSA aneurysms has involved both surgical and endovascular methods. Vessel sacrifice is usually required using either approach. Endovascular treatment of LSA aneurysms has involved glue embolization of the involved artery [7]. However, endovascular catheterization and treatment is often difficult because of the small caliber of the vessels involved and the often acute angle of origin from MCA trunk. Surgical approaches, which usually involve trapping of the aneurysm and vessel occlusion, are difficult because of the common distal location in and around the basal ganglia as well as the close proximity of other perforating arteries. As a result, stereotactic guidance with either CTA or three-dimensional angiography is often used to aid in the localization of such small and deep lesions [9,10]. As shown in this case, however, these techniques can be inaccurate, especially for lateral perforators coursing medially along the M1 segment. Thus, we feel superselective angiography is indicated in these cases. This provides both an option for endovascular therapy as well as more accurate delineation for surgical planning for these rare aneurysms.

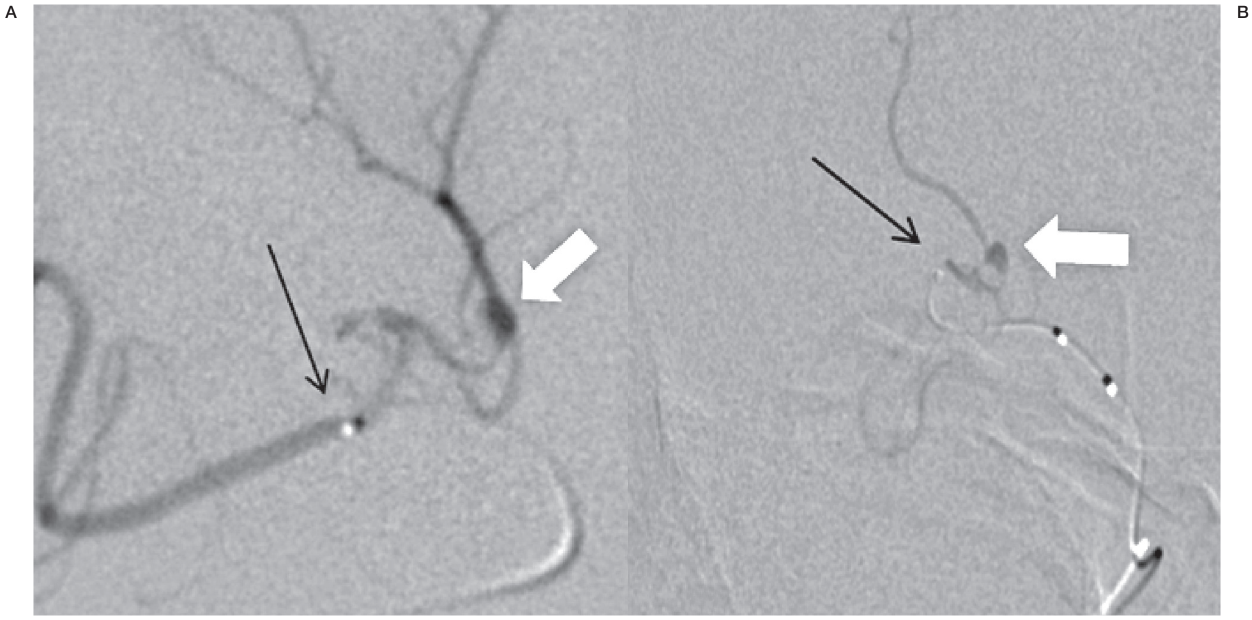

Figure 2 A) Superselective M2 injection (black arrow) demonstrates that the aneurysm actually arising from a M2 lenticulostriate branch. Thick white arrow shows a fusiform aneurysm. B) Lateral projection of super selective microcatheter run from the ostia of lenticulostriate artery (black arrow) arising from an M2 MCA branch reveals the small fusiform aneurysm (thick white arrow).

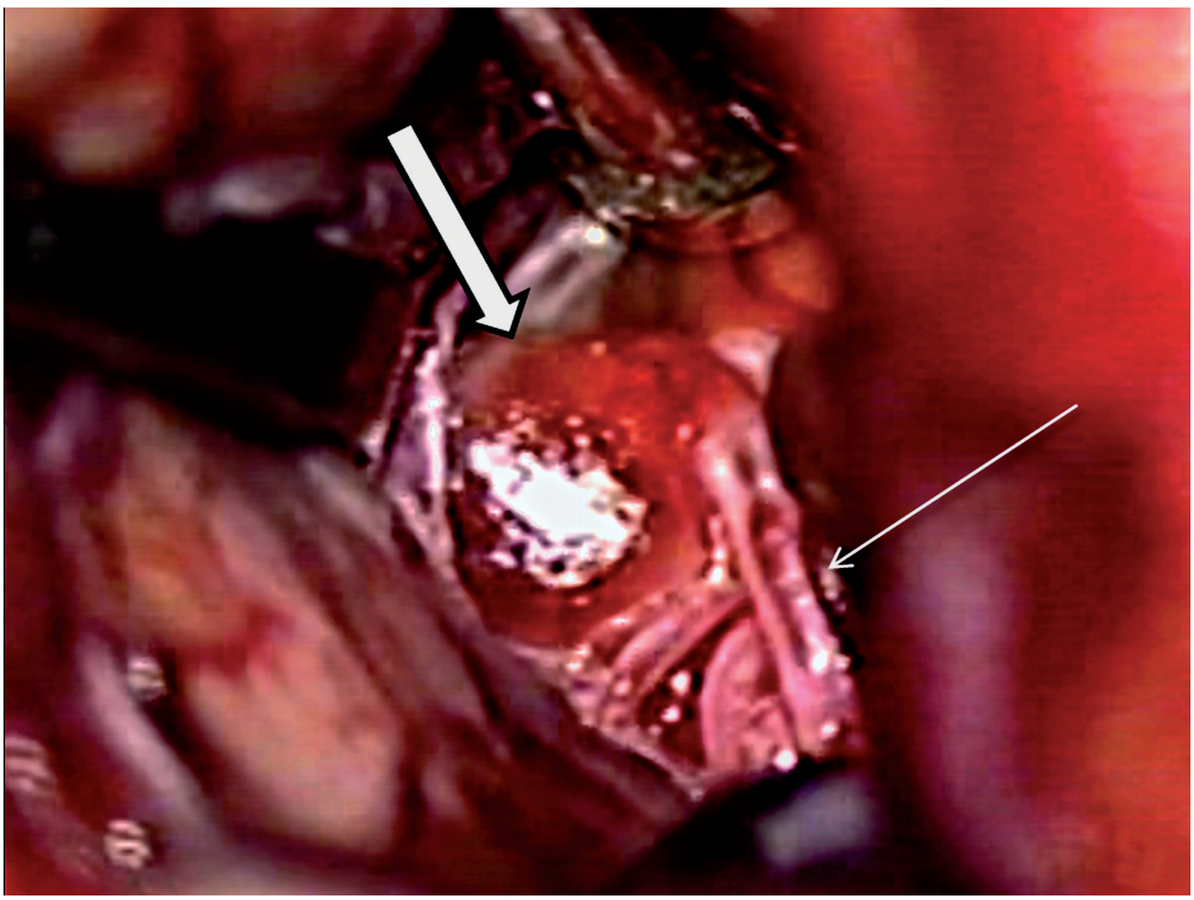

Figure 3 Intraoperative image reveals parent LSA (thin white arrow) with tiny 2 mm LSA aneurysm (thick white arrow).

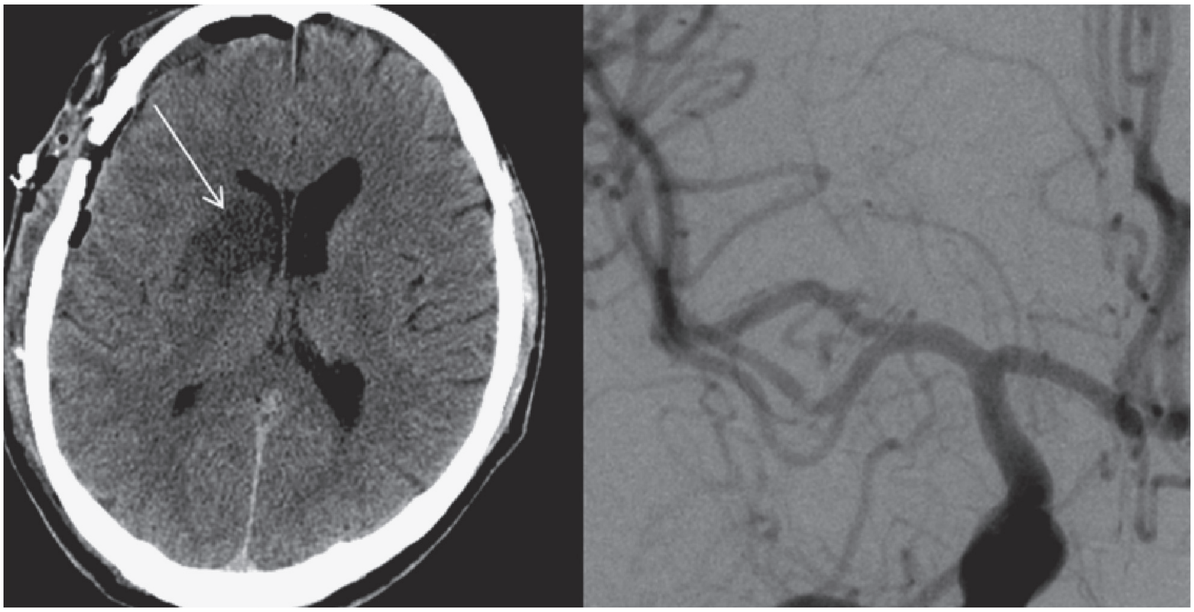

Figure 4 A) Post operative NCCT reveal a hypodensity in the right basal ganglia (white arrow) consistent with a small infarct. B) DSA post clipping shows no residual aneurysm.

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